# A common surgical emergency complicated by anterior spinal cord infarction

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DECLARATIONS

**Competing interests**
None declared

**Funding**
None

**Ethical approval**
Written consent to publication was obtained from the patient or next of kin

**Guarantor**
AW

**Contributorship**
Both authors contributed equally

**Acknowledgements**
None

**Reviewer**
Ahmed-Ramadan Sadek

This case explores how the presentation of a perforated duodenal ulcer is masked by a preceding anterior spinal artery infarction.

## Case report

A 75-year-old man was admitted to Accident and Emergency, drowsy, hypoglycaemic and unable to move his legs. He was found early in the morning by a family member; the last thing he remembered was watching television while sitting in a chair the night before. He was previously fully mobile, with a past medical history including insulin-dependent type 2 diabetes mellitus, hypercholesterolemia, hypertension and an alcohol drinking history of 30 units per week for 30 years. He reported no symptoms suggestive of either gastro-oesophageal or peptic ulcer disease and had no medical history of gastrointestinal disease.

On examination of his neurological system his cranial nerves were intact, upper limb power, reflexes, coordination and sensation were all normal. However on examination of his lower limbs, he had marked weakness predominantly affecting his proximal muscles, particularly on the right side (R > L 3/5 / 2/5). He had absent knee and ankles jerks, and plantar reflexes were up-going bilaterally. On sensory examination the spinothalamic tract was absent below the level of T6–T7 but the dorsal columns were unaffected. He was incontinent in both bladder and bowel function.

As spinal cord pathology of acute onset was suspected, urgent magnetic resonance imaging (MRI) of the spine was performed on day 1 (Figure 1). This demonstrated a T5–T8 anterior infarction of his spinal cord; no compressive or haemorrhagic lesions were identified. Urgent neurosurgical and neurological advice was sought following which medical management with Aspirin, blood pressure lowering medication and prophylaxtic dose low molecular weight heparin was commenced on day 2. During the following days he underwent early neurorehabilitation with physiotherapy.

On day 4 he was awoken with nausea and an episode of dark brown vomit. On examination the abdomen was soft with mild distension. In addition he had absent bowel sounds and bilateral shoulder tip pain. Of note there was no guarding or rebound tenderness.

Initial impression was an upper GI bleed and an emergency endoscopy was booked as well as an abdominal X-ray and erect chest X-ray. The chest X-ray showed bilateral signs of air under the diaphragm and the patient was urgently referred to the general surgeons with a suspected gastrointestinal perforation (Figure 2).

Confirmation of the perforation was sought by way of a computerized tomography (CT) scan of the abdomen. Urgent laparoscopic surgical management of the perforated viscus was subsequently organized.

Postoperatively his rehabilitation course was slow. Over the following 4 weeks he regained some function in his legs with the proximal muscles regaining more than distal groups (R > L 4/5 / 3/5) though he remained incontinent of urine and faeces. He was discharged to a rehabilitation centre with a long-term catheter.

## Discussion

Spinal cord infarction and its ensuing clinical and pathological pattern is a relatively rare disorder that has been shown to account for approximately 1% of all strokes.[1] Studies have demonstrated that cord infarctions, like cerebral infarctions, result from a multitude of aetiologies that interrupt the vascular blood flow. They fall into two distinct

**Figure 1**
**Sagittal MRI cervical and thoracic spine from admission. This demonstrates an area of infraction in the anterior spinal cord at the T5–T8 region, the location of which is indicated by the white arrow**

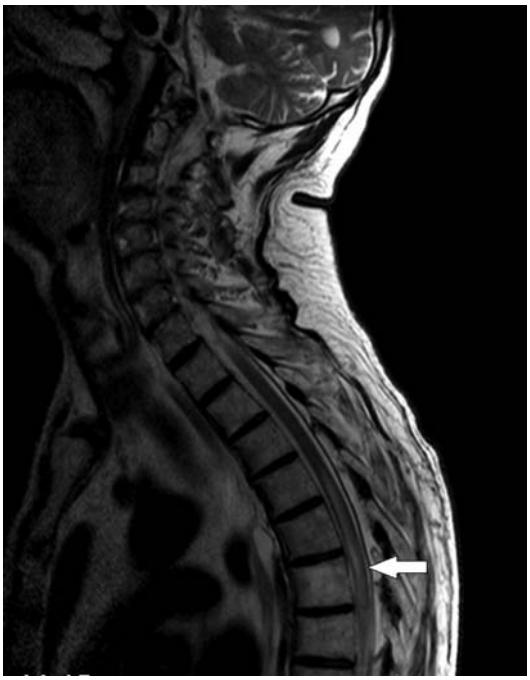
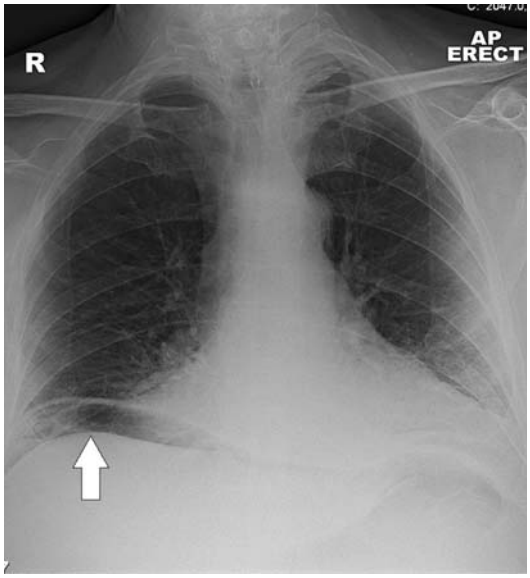


**Figure 2**
**Frontal chest radiograph performed on day 4 demonstrating air under the diaphragm bilaterally. This is particularly clear beneath the right diaphragm as indicated by the white arrow**




clinical patterns: regional interruptions of blood flow, which can occur for example during aortic aneurysm rupture and surgical repair, these will cause a more general transverse spinal cord ischaemia. Second, local interruption of spinal vasculature and blood flow, such as during an anterior spinal cord artery embolism, will cause ischaemia in only its affected territory. The clinical picture associated with anterior spinal artery infarction, as presented in this case, where bilateral motor and spinothalamic deficits ensue, has been shown to be the most common.

Anterior spinal cord infarction can affect the spinothalamic tracts, spinocerebellar tracts, and corticospinal tracts, as well as the afferent and efferent fibres exiting the dorsal root at that level. These neurones locate to the region supplied by the anterior spinal artery. This artery is particularly vulnerable in the T4–T8 region where it is supplied by the radicular artery (the artery of Adamkiewicz) which has poor anastomoses and is effectively an end artery. Indeed the lower thoracic region, particularly around segments T8 and T9, is the most frequently affected.[2] In this syndrome the posterior columns, which are primarily supplied by the posterior spinal arteries (these are well anastomosed) are spared.

We have described a classical case of thoracic anterior spinal cord infarction, with upper motor neurone lesion signs including paraparesis, extensor plantar response, incontinence and loss of spinothalamic sensation with preservation of dorsal column sensation. It is also important to recognize that a lower motor neurone lesion will affect the segmental level, in this case T5–T8, with signs including weakness, fasciculation and wasting, and loss of all sensory modalities.

The classical presentation of perforated peptic ulcer disease is that of sudden onset of epigastric pain, which becomes more generalized. Vomiting of brownish or blood stained fluids may also be seen.

On examination there are signs of a chemical peritonitis with abdominal tenderness and guarding or 'board-like-rigidity'. Diminished bowel sounds and low grade fever are often present.

Signs of bacterial peritonitis, such as hypotension and fever are later signs which typically appear at least 24 h following the perforation. A plain erect chest X-ray showing gas beneath the diaphragm confirms the presence of a perforation. A contrast gastroduodenogram or CT scan of the abdomen can confirm the presence and location of a persistent leak. Emergency surgery for peritoneal washout and closure of the perforation is strongly indicated in the management of a leaking perforated duodenal ulcer.[3,4]

Our case study is striking in that it lacked many of the classical signs and symptoms associated with this pathology. It is reasonable to propose that the symptoms of pain were masked by the underlying spinal cord lesion as it affected the region of the spinal cord thought to mediate noxious stimuli from the duodenum.

The duodenum is richly innervated by both parasympathetic and sympathetic afferent nerve fibres. Parasympathetic fibres project via the vagus nerve are generally believed to carry information regarding digestion. Splanchic sympathetic fibres project to the spinal cord particularly around the lower thoracic and upper lumbar region; T9-L1 in a variety of mammalian species. Duodenal afferents projecting via the splanchic nerve and celiac plexus to T9–T10 have been shown to respond to noxious stimuli in rats.[5,6] It follows that the lesion affecting spinal cord segments T5–T8 in our case effectively anaesthetized the duodenal viscera, thus preventing our patient from feeling pain following duodenal perforation.

In addition, the motor sensation to the abdominal musculature, via the lower thoracovertebral nerves, has segmental origins between T7 and T12. A lesion affecting the lower motor neurones in these segments, such as that presented here, could result in weakness and arreflexia of the abdominal muscles. This accounts for the absence of guarding and board like rigidity in this case.

We were alerted to the possibility of potentially serious abdominal pathology by the onset and persistence of bilateral shoulder pain. The diaphragm is known to be innervated by the phrenic nerve which originates from spinal segments C3–C5. This nerve receives afferent fibres, which innervate the central tendon of the diaphragm. Irritation to the diaphragm, by for example a large collection of air beneath the diaphragm, results in the clinical phenomenon of referred pain in the C3–C5 somatic regions. This anatomical curiosity was of great clinical importance; the sparing of visceral sensation at the diaphragm by its segmental origin rostral to our lesion was the major clue of the underlying pathology. Indeed as Figure 2 demonstrates, there was a large amount of free air in the subdiaphragmatic region bilaterally.

Our case corroborates evidence that noxious stimuli to the duodenum are mediated via spinal segments in the lower thoracic region in humans. It is a unique case which demonstrates the impact that spinal cord pathology can have on the presentation of seemingly unconnected pathology such as duodenal perforation. By recognizing the significance of subtle abdominal signs in the context of what was effectively a spinal anaesthesia, we were able to offer our patient appropriate, timely surgical management and avoided the potentially catastrophic onset of a bacterial peritonitis.

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